# Immune response and serum immunoglobulin G concentrations in beef calves suckling cows of differing body condition score at parturition and supplemented with high-linoleate or high-oleate safflower seeds[1]

**S. L. Lake,*[2] E. J. Scholljegerdes,*[3] W. T. Small,*[4] E. L. Belden,† S. I. Paisley,* D. C. Rule,* and B. W. Hess*[5]**

*Department of Animal Science and †Department of Veterinary Sciences, University of Wyoming, Laramie 82071-3684

**ABSTRACT:** Two experiments were conducted to determine the effect of maternal lipid supplementation on the immune response to antigenic challenge in suckling calves. In Exp. 1, beginning 1 d postpartum, 18 primiparous crossbred beef cows were fed Foxtail millet hay and a low-fat (control) supplement or a supplement containing cracked, high-linoleate safflower seed in individual feeding stanchions until d 40 of lactation. The diets were formulated to provide similar quantities of N and TDN, and the linoleate diet was formulated to contain 5% of DMI as fat. Calves were injected s.c. with 15 mg of antigen (ovalbumin) at d 21 and again at d 35 of age. To measure the total serum antibody production in response to the antigen, blood samples were collected from the calves every 7 d via jugular venipuncture from d 14 to 42. Calves from linoleate-supplemented cows had a decrease ($P = 0.04$) in total antibody production in response to ovalbumin and appeared to have a delayed response to antigen challenge. Total antibody production increased ($P < 0.001$) after secondary exposure to ovalbumin. In Exp. 2, 36 Angus × Gelbvieh beef cows that were nutritionally managed to achieve a BCS of 4 or 6 at parturition were used to determine the effects of prepartum energy balance and postpartum lipid supplementation on the passive transfer of immunoglobulins and the immune response to antigenic challenge in their calves. Beginning at 3 d postpartum and continuing until d 60 of lactation, cows were fed hay and a low-fat control supplement or supplements consisting of either cracked, high-linoleate or high-oleate safflower seeds. Safflower seed supplements were formulated to provide 5% of DMI as fat. Calves were injected s.c. with 15 mg of ovalbumin at 21 d of age and again at 48 d of age. The antibody responses were determined in serum; cell-mediated immunity was assessed by intradermal antigen injection at 60 d of age. A trend was noted ($P = 0.10$) for calves suckling control-supplemented cows to have a greater response to antigen compared with calves from linoleate- and oleate-supplemented cows; however, no difference was observed among treatments ($P = 0.86$) in cell-mediated immune response. Postpartum oilseed supplementation in beef cows appears to decrease antibody production in response to antigenic challenge in suckling calves. However, BCS at parturition did not influence passive transfer of immunoglobulins in neonatal calves.

**Key words:** beef calf, immune response, lipid supplementation, passive transfer

 J. Anim. Sci. 2006. 84:997–1003

## INTRODUCTION

Dietary lipid supplementation is often used to meet nutritional demands associated with lactation and reproduction (Funston, 2004; Hess et al., 2005). Incorporation of fatty acids into membrane phospholipids is dependent on fatty acid availability (De Pablo and De Cienfuegos, 2000). Moussa et al. (2000) reported that fatty acid profiles of spleen lymphocytes in rats were reflective of dietary fatty acids. The specific roles that PUFA have in the regulation of cellular function (Calder et al., 2002) suggest that altering the fatty acid profile of lymphocyte membrane phospholipids may affect immune response.

Poor prepartum nutritional management has been associated with detrimental effects on postpartum calf immune response, possibly because of dystocia (Quigley

[1]This project was supported by National Research Initiative Competitive Grant no. 2002-35206-11632 from the USDA Cooperative State Research, Education, and Extension Service.

[2]Current address: Department of Animal Science, Purdue Univ., West Lafayette, IN 47907.

[3]Current address: USDA-ARS, NGPRL, Mandan, ND 58554.

[4]Current address: Agri Beef Co., American Falls, ID 83211.

[5]Corresponding author: brethess@uwyo.edu

Received July 7, 2005.

Accepted November 21, 2005.

**Table 1.** Composition of experimental diets fed to primiparous beef cows (Exp. 1)[1]

| Item | Control | Linoleate |
|---|---|---|
| Ingredient | (% of DM) | |
| Foxtail millet hay | 85.2 | 89.9 |
| Cracked corn | 9.5 | — |
| Safflower seed meal | 4.7 | — |
| Cracked, high-linoleate safflower seeds | — | 9.5 |
| Molasses | 0.55 | 0.61 |
| Analyzed composition | | |
| CP | 11.6 | 11.6 |
| IVDMD | 59.5 | 55.7 |
| Crude fat | 2.5 | 5.4 |
| Fatty acid | (wt %) | |
| 16:0 | 15.1 | 6.1 |
| 18:1 | 20.0 | 14.2 |
| 18:2 | 25.1 | 67.3 |
| 18:3 | 4.0 | 0.7 |

[1]Diets were formulated to be isocaloric and isonitrogenous and to meet the energy requirements of a 410-kg primiparous beef heifer producing 9 kg of milk during peak lactation.

and Drewry, 1998) or decreased immunoglobulin G (**IgG**) absorption (Hough et al., 1990). Immune status of neonatal calves has a profound effect on the life-long health status of beef cattle (Wittum and Perino, 1995). Wittum and Perino (1995) indicated that calves with lower levels of IgG and total plasma protein at 24 h postpartum had greater incidences of preweaning and feedlot morbidity and mortality, which are often considered the greatest determinants of profitability in feedlot cattle (Gardner et al., 1999).

To our knowledge, the effect of maternal lipid supplementation on suckling beef calf immune response has not been investigated. We hypothesized that maternal prepartum nutritional management and postpartum lipid supplementation of the dam would influence immune response in suckling calves. Our objectives were to evaluate BCS at parturition and supplementation of cows with cracked high-linoleate or high-oleate safflower seeds on calf immune response to antigenic challenge.

## MATERIALS AND METHODS

### *Exp. 1*

***Animals and Diets.*** All animal care, handling techniques, and sample collection protocols were approved by the University of Wyoming Institutional Animal Care and Use Committee. Starting on d 1 postpartum, primiparous, fall-calving, 2-yr-old Angus beef cows [n = 18; initial BW = 409 ± 24.2 kg; BCS = 5.25 (1 = emaciated to 9 = obese; Wagner et al., 1988)] were housed in a drylot.

The cows were fed Foxtail millet hay and were randomly assigned to be fed either a low-fat control or a cracked, high-linoleate safflower seed supplement (Table 1) in individual feeding stanchions until d 40 of lactation. Previous research at the University of Wyoming reported that cows of similar genetics produced 9 kg of milk during peak lactation (Bottger et al., 2002). Therefore, the diets were formulated to meet the nutrient requirements for a primiparous, 410-kg beef cow producing 9 kg of milk at peak lactation (NRC, 2000). The diets were also formulated to be isonitrogenous and isocaloric; the high-linoleate diet was formulated to provide 5% of DMI as fat. The dietary ingredients were analyzed for CP (Leco FP-528, Leco Corp., St. Joseph, MO), IVDMD (Daisy II Incubator, Ankom Tech. Corp., Fairport, NY), crude fat (2050 Soxtec Avanti Auto Control Unit, Foss Tecator, Eden Prairie, MN), and fatty acids via direct transesterification (Whitney et al., 1999) with methanolic HCl (Kucuk et al., 2001).

To assess calf performance, calf BW was recorded on 2 consecutive days on d 1 and 40 of age, and the average of these weights was used. At 21 and 35 d of age, the calves received a s.c. injection of 15 mg of ovalbumin (Sigma-Aldrich, St. Louis, MO) suspended in 2 mL of 10% (wt/vol) potassium alum (Mallinckrodt Baker, Inc. Phillipsburg, NJ) in physiological saline.

***Sample Collection and Analysis.*** Every 7 d from 14 to 62 d of age, rectal temperatures were recorded, and blood samples were collected via jugular venipuncture to measure antibody production in response to antigen challenge. Serum was harvested from whole blood after clotting at 4°C for 8 h and was centrifuged at 500 × *g* for 20 min. Serum was stored at –20°C until the analysis of antiovalbumin antibodies using indirect ELISA (Coligan et al., 1991).

Serum was thawed overnight and mixed with serum conjugate diluent (75 m*M* NaCl, 0.62 m*M* EDTA, 25 m*M* Tris base, and 2% fetal bovine serum) to create a 1:40 dilution. Fifty microliters of ovalbumin carbon-coating buffer [0.1 g of ovalbumin/100 mL of carbon-coating buffer (34 m*M* sodium carbonate and 15 m*M* anhydrous sodium carbonate)] was used to coat each well of a 96-well plate (Immulon 2, Dynex Tech., Chantilly, VA) at 4°C for 24 h. The plates were rinsed 3 times with Tween buffer solution (0.05 mL/L of Tween 20; 0.14 *M* NaCl; 2.7 m*M* KCl; and 8.4 m*M* sodium phosphate) and patted dry. The plates were incubated with 100 μL of 10% fetal bovine serum carbon-coating buffer (10 g of fetal bovine serum/mL; 34 m*M* sodium carbonate and 15 m*M* anhydrous sodium carbonate), incubated for 2 h at room temperature, and subsequently rinsed 3 times as just described. Serum samples were added (50 μL) to each well and incubated at 37°C for 1 h. The plates were rinsed 3 times as previously described and incubated with 50 μL of rabbit antibovine IgG-horseradish peroxidase conjugate for 30 min at 37°C. After rinsing 3 times as described, 50 μL of enzyme substrate [6 mL of 0.1 *M* sodium acetate; 40 μL of 0.11 *M* citric acid; 63 μL of tetramethylbenzidine solution (100 mg of tetramethylbenzidine/10 mL of dimethyl sulfoxide), and 0.97 μL of $H_2O_2$] was allowed to develop for 3 to 4 min on a rocker platform (MicroShaker II, Dynatech Laboratories Inc., Chantilly, VA), and the

reaction was terminated with the addition of 50 μL of 2 *M* $H_2SO_4$. Total antibody production in response to ovalbumin challenge was determined by a dual absorbance (490 and 595 nm) microplate reader (model 3550 Microplate Reader, Bio-Rad, Hercules, CA). Data presented represent absorbance of total ovalbumin-specific antibodies (inter- and intraassay CV = 8.1 and 5.4, respectively).

***Statistical Analysis.*** All data were analyzed by ANOVA using the MIXED procedures of SAS (SAS Inst., Inc., Cary, NC). The model included effects of maternal dietary treatment, day of age, and treatment × day of age. Individual calf identification was used to specify variation between animals using the RANDOM statement, and day of age was used as the repeated effect. Using likelihood ratio testing, an autoregressive order one structure was deemed most appropriate for within-subject effects (the effects associated with day of sampling).

## *Exp. 2*

***Animals and Diets.*** Cows were managed as described by Lake et al. (2005). Briefly, in a 2-yr experiment (n = 36/yr), spring-calving, 3-yr-old Angus × Gelbvieh beef cows (n = 72) were managed nutritionally to achieve a BCS (1 = emaciated to 9 = obese; Wagner et al., 1988) of 4 ± 0.07 (initial BW = 479.3 ± 36.3 kg) or 6 ± 0.07 (initial BW = 579.6 ± 53.1 kg) at parturition. Beginning at 3 d postpartum, cows were placed into 1 of 6 drylot pens (6 cows per pen) with individual feeding stanchions and were fed twice daily. Cows were fed diets of hay (2.13% of BW during yr 1 and 2.03% of BW during yr 2) plus a low-fat control supplement (0.57% of BW during yr 1 and 0.30% of BW during yr 2) or supplements with either high-linoleate, cracked safflower seeds (hay at 2.32% of BW and supplement at 0.39% of BW during yr 1; hay at 2.03% of BW and supplement at 0.23% of BW during yr 2) or high-oleate, cracked safflower seeds (hay at 2.32% of BW and supplement at 0.40% of BW during yr 1; hay at 2.03% of BW and supplement at 0.24% of BW during yr 2) until d 60 of lactation. The diets (Table 2) were formulated to meet the energy requirements of a 544-kg beef cow producing 9 kg of milk at peak lactation. The diets were formulated to be isonitrogenous and isocaloric; lipid-supplemented diets were formulated to be isolipidic, providing 5% of DMI as fat. The dietary ingredients were analyzed for CP, crude fat, and fatty acids as described previously.

The cows were monitored for signs of dystocia by trained University of Wyoming personnel to ensure that suckling occurred, and cow-calf pairs were monitored after calving. Calf performance was reported by Lake et al. (2005). At 21 and 48 d of age, the calves were injected s.c. with 15 mg of ovalbumin and suspended in 2 mL of potassium alum. At d 101, the calves were weaned. At 35 and 50 d postweaning, calves were injected s.c. with 15 mg of ovalbumin suspended in 2 mL of potassium alum.

**Table 2.** Composition of diets consumed by lactating beef cows (Exp. 2)[1]

| | Diet | | |
|---|---|---|---|
| Item | Control | Linoleate | Oleate |
| Ingredient | | % of DM | |
| Foxtail millet hay | 87.2 | 89.7 | 89.6 |
| High-linoleate safflower seed | — | 8.1 | — |
| High-oleate safflower seed | — | — | 7.6 |
| Soybean meal | 0.6 | — | 0.6 |
| Molasses | 0.6 | 0.6 | 0.6 |
| Beet pulp pellets | 10.0 | — | — |
| Minerals | 1.6 | 1.6 | 1.6 |
| Analyzed composition | | | |
| CP | 11.2 | 11.4 | 11.4 |
| TDN[2] | 69.7 | 70.1 | 70.1 |
| Crude fat | 2.2 | 5.0 | 5.0 |
| Fatty acid | | wt % | |
| 16:0 | 19.8 | 10.0 | 8.0 |
| 18:0 | 2.7 | 3.2 | 0.2 |
| 18:1 | 10.4 | 10.3 | 71.3 |
| 18:2 | 22.4 | 68.1 | 10.9 |
| 18:3 | 1.7 | 0.4 | 0.6 |

[1]Diets were formulated to be isocaloric and isonitrogenous and to meet the energy requirements of a 544-kg beef cow producing 9 kg of milk during peak lactation. Lipid-supplemented diets were formulated to provide 5% of DMI as fat.

[2]The TDN for the hay samples was estimated from ADF values (Linn and Martin, 1989), whereas tabular values (NRC, 1982) were used to calculate the TDN of the supplemental ingredients.

***Sample Collection and Analysis.*** Blood samples were collected via jugular venipuncture at 48 h after birth for analysis of serum IgG concentrations using a commercially available antigen trap ELISA kit (Bethyl Laboratories, Montgomery, TX; intraassay CV = 4.8%). Beginning at 14 d of age, blood samples were taken via jugular venipuncture every 3 d until d 63 postpartum and also on d 35, 42, and 48 and on d 54 postweaning. Serum was harvested and stored at −20°C until analyzed for total antibodies produced against ovalbumin using indirect ELISA (intraassay CV = 7.8%) as described previously. Cell-mediated immunity was assessed at 57 d of age by a 100-μL intradermal antigen injection of 1 mg of ovalbumin suspended in 0.01 *M* phosphate buffer diluted in physiological saline. The response to the intradermal injection was determined at 48 and 72 h (on 59 and 60 d of age) by measuring the diameter of the nodule formed around the injection site with calipers.

***Statistical Analysis.*** The effect of maternal BCS at parturition on serum IgG concentrations at 48 h was analyzed by a one-way ANOVA with the GLM procedure of SAS because maternal postpartum dietary treatment did not begin until after serum samples were collected. The preweaning serum antibody concentration and the cell-mediated response were analyzed as repeated measures with a 2 × 3 arrangement of treatments in a randomized complete block design using the

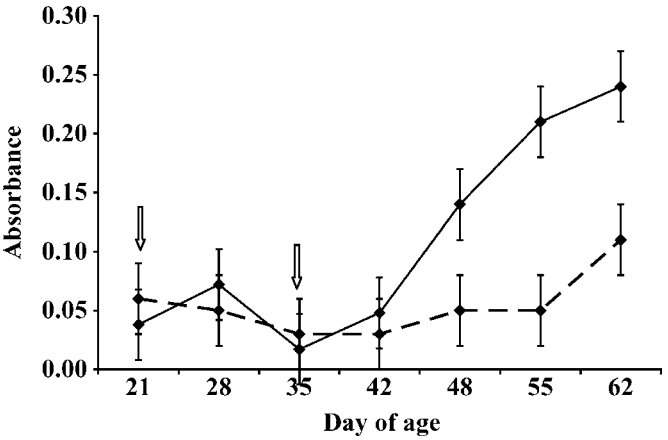


**Figure 1.** A maternal dietary treatment × day of sampling interaction was detected ($P = 0.05$) for total antibodies produced against ovalbumin (Exp. 1). The solid line represents calves suckling control-supplemented cows, and the dashed line represents calves suckling cows fed the high-linoleate supplement. Calves were injected s.c. with 15 mg of ovalbumin suspended in 2 mL of potassium alum at d 21 and again at d 35 of age (indicated by the arrows).

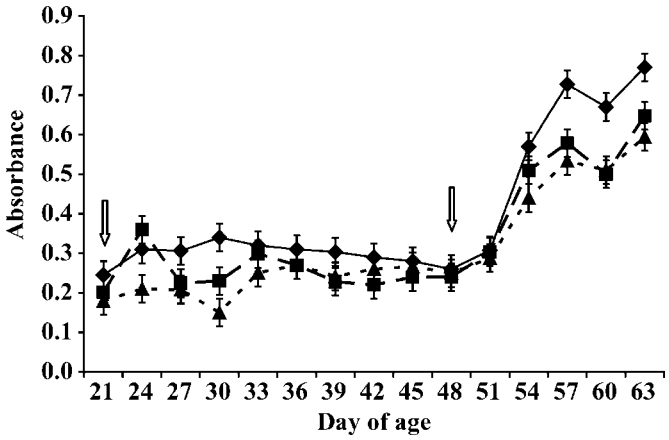


**Figure 2.** No maternal dietary treatment × day of sampling interaction was detected ($P = 0.55$) for total antibodies produced against ovalbumin in preweaned calves through 63 d of age (Exp. 2). Immunoglobulin production increased subsequent to ovalbumin challenge at the secondary exposure during the first 63 d of age ($P < 0.001$). The solid lines represent calves suckling control-supplemented cows, the large dashed lines represent calves suckling linoleate-supplemented cows, and the small dashed lines represent calves suckling oleate-supplemented cows. Calves were injected s.c. with 15 mg of ovalbumin suspended in 2 mL of potassium alum at 21 and 48 d of age (indicated by the arrows).

MIXED procedures of SAS. The model included the effects of BCS at parturition, maternal dietary treatment, day of age, and all possible interactions. Individual calf identification was used to specify the variation between animals using the RANDOM statement, and day of age was used as the repeated effect. Postweaning serum antibody concentrations were analyzed using the same model described for the preweaning data, but these data were analyzed separately to evaluate potential residual effects from maternal lipid supplementation during early lactation. Using likelihood ratio testing, an autoregressive order one structure was deemed most appropriate for the within-subject effects (the effects associated with days of age; Littell et al., 2000). No interactions were identified ($P = 0.55$ to 0.99); therefore, only main effects are presented. One cow-calf pair was removed from the study because of the death of the calf. Necropsy performed at the Wyoming State Veterinary Laboratory revealed that the calf's death was due to complications not attributed to the study; consequently, comparisons of main effects and interactions were determined using least square means.

## RESULTS

### *Exp. 1*

A maternal dietary treatment × day of sampling interaction was detected ($P = 0.05$) for total antibodies produced against ovalbumin (Figure 1). Calves from oleate-supplemented and linoleate-supplemented cows had decreased ($P = 0.04$) total antibody production in response to ovalbumin throughout the course of the experiment and appeared to have a delayed response to antigen challenge compared with control calves. Total antibody production against antigen challenge increased ($P < 0.001$) after secondary exposure to ovalbumin. No difference ($P = 0.90$; data not shown) was noted for rectal temperature throughout the study. Similarly, ADG did not differ ($P = 0.13$) in control calves compared with linoleate calves (0.66 vs. 0.90 ± 0.11 kg/d, respectively).

### *Exp. 2*

A maternal dietary treatment × day of sampling interaction was not detected ($P = 0.55$) for production of antibodies against ovalbumin (Figure 2). The main effects of BCS at parturition and maternal postpartum supplementation are presented in Table 3. Maternal BCS at parturition had no effect ($P = 0.52$) on serum IgG concentration from calves 48 h after birth. Maternal BCS at parturition did not influence calf production of antibody against antigen challenge in response to ovalbumin during the first 63 d of age ($P = 0.92$) or at weaning ($P = 0.99$). Calves suckling cows fed the control diet tended ($P = 0.10$) to have greater antibody production against ovalbumin through the first 63 d of age compared with calves suckling lipid-supplemented cows. However, no differences ($P = 0.80$) among treatments were detected in antibody production postweaning. Antibody production increased in all calves subse-

**Table 3.** Main effects of maternal BCS at parturition and postpartum dietary treatment on pre- and postweaning immune variables of calves (Exp. 2)

| | BCS | | Diet[1] | | | | *P*-value | |
|---|---|---|---|---|---|---|---|---|
| Item | 4 | 6 | Control | Linoleate | Oleate | SEM[2] | BCS | Treatment |
| n | 18 | 17 | 12 | 11 | 12 | — | — | — |
| 48-h serum IgG,[3] mg/mL | 15.6 | 13.4 | — | — | — | 2.4 | 0.52 | — |
| Preweaning antibodies[4] | 0.38 | 0.38 | 0.44 | 0.37 | 0.34 | 0.04 | 0.92 | 0.10 |
| Postweaning antibodies[5] | 0.37 | 0.37 | 0.37 | 0.36 | 0.38 | 0.03 | 0.99 | 0.76 |
| Cell-mediated immunity[6] | 0.55 | 0.56 | 0.61 | 0.53 | 0.52 | 0.08 | 0.90 | 0.62 |

[1]Refer to Table 2 for a description of the diet.
[2]Greatest SEM are presented.
[3]Blood samples were collected via jugular venipuncture at 48 h for analysis of serum immunoglubulin G (IgG) concentration.
[4]Total antibody production in response to ovalbumin challenge was determined by ELISA; the data represent the absorbance value for total ovalbumin-specific antibodies. Calves were injected s.c. with 15 mg of ovalbumin suspended in 2 mL of potassium alum at 21 and 48 d of age. Beginning on d 14 of age, blood samples were taken via jugular venipuncture every 3 d until d 48.
[5]Total antibody production in response to ovalbumin challenge was determined by ELISA; data represent the absorbance value for total ovalbumin-specific antibodies. Calves were weaned at 101 d of age and were given a 15-mg s.c. injection of ovalbumin suspended in 2 mL of potassium alum at d 35 and 50 postweaning. Blood samples were taken via jugular venipuncture on d 35, 42, 48, and 54 postweaning.
[6]Cell-mediated immunity was assessed by intradermal antigen injection (1 mg of ovalbumin) at 57 d of age; the diameter of the resulting skin wheal was measured in centimeters with calipers on d 59 and 60.

quent to secondary exposure to ovalbumin challenge during the first 63 d of age ($P < 0.001$) and at weaning ($P < 0.001$; data not shown). Maternal postpartum dietary treatment ($P = 0.62$) or BCS at parturition ($P = 0.90$) did not affect calf cell-mediated immunity at 60 d of age. Similarly, no differences were reported in calf ADG because of maternal BCS at parturition or maternal lipid supplementation (Lake et al., 2005).

## DISCUSSION

Fatty acids have important regulatory roles in cellular function and may potentially alter signal transduction (Calder et al., 2002). For example, saturated fatty acids, primarily myristic and palmitic acid, are covalently attached to membrane proteins and are believed to be involved with regulating intracellular signaling (Calder, 1996). Additionally, antibody production and secretion in response to antigen stimulation can be modulated by fatty acids (Pompéia et al., 2000). For example, inclusion of linoleic acid in the diets of mice impaired production of antibodies, including IgG after antigenic challenge (Pompéia et al., 2000). Incorporation of fatty acids into membrane phospholipids are dependent on availability (De Pablo and De Cienfuego, 2000), and fatty acid profiles of lymphocytes are reflective of the diet (Clamp et al., 1997; Moussa et al., 2000). Calf adipose tissue fatty acid profile in the current study was reflective of changes in milk fatty acid profile of cows consuming dietary lipid supplements (Lake et al., 2004). Thus, for the calf, intake of dietary fatty acids might have been sufficient to change lymphocyte fatty acid profile to reflect that of the calf's diet. Therefore, presumed alterations in fatty acid composition of lymphocyte membrane phospholipids might have resulted in decreased antibody production to antigenic challenge in calves suckling linoleate and oleate-supplemented cows.

The efficiency of the immune response relies not only on specific recognition of foreign antigens, but also on the amplification of lymphocyte proliferation. Unsaturated fatty acids can inhibit cell proliferation by an eicosanoid-independent action (Pompéia et al., 2000). Additionally, high concentrations of unsaturated fatty acids can cause apoptosis or necrosis through rapid loss of membrane integrity, lysosomal leakage, or cellular swelling (Pompéia et al., 2000). Therefore, changes in phospholipid fatty acid composition may alter the dynamics of membrane fluidity and plasma membrane characteristics that ultimately affect the immune system, which has been observed with macrophage function (De Pablo and De Cienfuegos, 2000). This may be attributed to changes produced in the activity of proteins associated with membranes acting as receptors or ion channels. Therefore, binding of cytokines to their respective receptors on lymphocyte membranes may be altered. Additionally, surface molecules such as adhesion and major histocompatibility molecules have been inhibited by PUFA (Hughes et al., 1996). Perhaps PUFA act as ionophores, resulting in an increased cellular maintenance requirement and decreased productivity of the lymphocyte. To our knowledge, literature is not available on suckling beef calf immune response to maternal dietary treatment. Nevertheless, decreased antibody production in calves suckling dams fed the linoleate supplement in response to ovalbumin challenge in Exp. 1 and 2 of the current study were consistent with results of research in rodents and may be attributed to alterations in membrane fluidity and/or lymphocyte proliferation. The lack of long-term effects suggests that antibody production was inhibited only during the time of lipid supplementation. Although no

long-term effects were detected in calf immune response to ovalbumin challenge, impeding immune function during the critical early stages of life may result in decreased survivability or performance and ultimately affect beef cattle production.

Calves are essentially born agammagluobulinemic because of the epitheliochoral placentation of ruminants (Mee et al., 1996). It has been established that some level of adaptive immunity develops by d 130 of gestation, but production of glucocorticoids associated with parturition effectively suppresses immunity in the neonatal calf (Barrington and Parish, 2001). Additionally, the ability of the newborn calf to mount an immune response to antigen is inefficient and is therefore considered immunonaive (Barrington and Parish, 2001).

The relationship between colostral IgG intake and serum IgG levels has been described as linear, and at any time, serum IgG levels are greater in calves consuming more IgG compared with calves consuming less IgG (Hopkins and Quigley, 1997). Therefore, the lack of difference in the current study in total IgG at 48 h suggests that cows at BCS 4 produced similar volumes of immunoglobulins and that calves of cows at BCS 4 absorbed colostral immunoglobulins similarly to calves born of cows at BCS 6. Our results are in agreement with Perino et al. (1995) who reported no differences in 24-h serum IgG concentration between calves born of cows ranging in BCS from 4 to 7. Burton et al. (1984) also reported no effects on immunoglobulin concentrations in the colostrum of cows fed restricted diets; however, absorption of immunoglobulins were reduced in calves from restricted cows. Similarly, Hough et al. (1990) noted no differences in IgG concentrations in the colostrum of beef cows fed diets at 100 or 57% of NRC requirements prepartum, demonstrating that intake level did not affect colostrum volume. However, calves from nutrient-restricted cows, as well as calves from nonnutrient-restricted cows had a 21% reduction in IgG absorption when fed colostrum from nutrient-restricted cows. Therefore, maternal prepartum nutrient restriction appears to influence neonatal calf immunoglobulin absorption. The decrease in immunoglobulin absorption of calves from nutrient-restricted cows did not appear to be associated with intestinal absorptive capabilities of the calf, but rather appeared to be due to alterations in constituents of the colostrum. Transfer of immunoglobulins from maternal serum to colostrum in cattle normally begins 4 wk before parturition and reaches maximum a few days before parturition (Olson et al., 1981). Lack of differences in immunoglobulin absorption in the current study is not surprising because cows were nutritionally managed to lose condition during the second trimester and were fed to meet protein requirements (Lake et al., 2005) during the third trimester when colostrum production and the majority of fetal development occurs. Therefore, previously reported diminished immunoglobulin absorption in calves of nutrient-restricted cows may depend on timing of nutrient restriction within the gestation cycle.

## IMPLICATIONS

Although maternal dietary lipid supplementation did not affect long-term immune response to ovalbumin, influencing immune function during the critical early stages of life may decrease productivity of the calf throughout its lifecycle. Beef cattle producers should be aware of potential effects of maternal supplementation on calf immune status. Results from the current study suggest that maternal body condition score at parturition did not affect passive transfer of immunoglobulins or immune response to antigen in calves. However, the relationship between prepartum nutritional status and transfer of passive immunity should be further studied to elucidate conflicting results in published data.

## LITERATURE CITED

Barrington, G. M., and S. M. Parish. 2001. Bovine neonatal immunology. Vet. Clin. North Am. Food Anim. Pract. 17:463–476.

Bottger, J. D., B. W. Hess, B. M. Alexander, D. L. Hixon, L. F. Woodard, R. N. Funston, D. M. Hallford, and G. E. Moss. 2002. Effects of supplementation with high linoleic or oleic cracked safflower seeds on postpartum reproduction and calf performance of primiparous beef heifers. J. Anim. Sci. 80:2023–2030.

Burton, J. H., A. A. Hosein, D. G. Grieve, and B. N. Wilkie. 1984. Immunoglobulin absorption in calves as influenced by dietary protein intakes of their dams. Can. J. Anim. Sci. 64(Suppl.):185. (Abstr.)

Calder, P. C. 1996. Effects of fatty acids and dietary lipids on cells of the immune system. Proc. Nutr. Soc. 55:127–150.

Calder, P. C., P. Yaqoob, F. Thies, F. A. Wallace, and E. A. Miles. 2002. Fatty acids and lymphocyte functions. Br. J. Nutr. 87:S31–S48.

Clamp, A. G., S. Ladha, D. C. Clark, R. F. Grimble, and E. K. Lund. 1997. The influence of dietary lipids on the composition and membrane fluidity of rat hepatocyte plasma membrane. Lipids 32:179–184.

Coligan, J. E., A. M. Kruisbeek, D. H. Margulies, E. M. Sherach, and W. Strober. 1991. Current Protocols in Immunology. John Wiley and Sons, Hoboken, NJ.

De Pablo, M. A., and G. A. De Cienfuegos. 2000. Modulatory effects of dietary lipids on immune system functions. Immunol. Cell Biol. 78:31–39.

Funston, R. N. 2004. Fat supplementation and reproduction in beef females. J. Anim. Sci. 82(E. Suppl.):E154–E161.

Gardner, B. A., H. G. Dolezal, L. K. Bryant, F. N. Owens, and R. A. Smith. 1999. Health of finishing steers: Effects on performance, carcass traits, and meat tenderness. J. Anim. Sci. 77:3168–3175.

Hess, B. W., S. L. Lake, E. J. Scholljegerdes, T. R. Weston, V. Nayigihugu, J. D. C. Molle, and G. E. Moss. 2005. Nutritional controls of beef cow reproduction. J. Anim. Sci. 83(E. Suppl.):E90–E106.

Hopkins, B. A., and J. D. Quigley. 1997. Effects of method of colostrum feeding and colostral supplementation on serum IgG concentrations in neonatal calves. J. Dairy Sci. 80:979–983.

Hough, R. L., F. D. McCarthy, H. D. Kent, D. E. Eversole, and M. L. Wahlberg. 1990. Influence of nutritional restriction during late gestation on production measures and passive immunity in beef cattle. J. Anim. Sci. 68:2622–2627.

Hughes, D. A., S. Southon, and A. C. Pinder. 1996. (n-3) polyunsaturated fatty acids modulate the expression of functionally associated molecules on human monocytes in vitro. J. Nutr. 126:603–610.

Kucuk, O., B. W. Hess, P. A. Ludden, and D. C. Rule. 2001. Effect of forage:concentrate ratio on ruminal digestion and duodenal flow of fatty acids in ewes. J. Anim. Sci. 79:2233–2240.

Lake, S. L., B. W. Hess, E. J. Scholljegerdes, R. L. Atkinson, and D. C. Rule. 2004. Milk and calf adipose tissue fatty acid changes in response to maternal supplementation with high-linoleate or high-oleate safflower seeds. J. Anim. Sci. 82(Suppl. 2):102. (Abstr.)

Lake, S. L., E. J. Scholljegerdes, R. L. Atkinson, V. Nayigihugu, S. I. Paisley, D. C. Rule, G. E. Moss, T. J. Robinson, and B. W. Hess. 2005. Body condition score at parturition and postpartum supplemental fat effects on cow and calf performance. J. Anim. Sci. 83:2908–2917.

Linn, J. G., and N. P. Martin. 1989. Forage quality tests and interpretation. Univ. Minnesota Ext. Ser. Publ. AG-FO-2637, St. Paul, MI.

Littell, R. C., J. Pendergast, and R. Natajan. 2000. Modeling covariance structure in the analysis of repeated measures data. Stat. Med. 19:1793–1819.

Mee, J. F., K. J. O'Farrell, P. Reitsma, and R. Mehra. 1996. Effect of whey protein concentrate used as a colostrum substitute or supplement on calf immunity, weight gain, and health. J. Dairy Sci. 79:886–894.

Moussa, M., J. Tkaczuk, J. Ragab, J. Garcia, M. Abbal, E. Ohayon, J. Ghisolfi, and J. P. Thouvenot. 2000. Relationship between the fatty acid composition of rat lymphocytes and immune function. Br. J. Nutr. 83:327–333.

NRC. 1982. United States-Canadian Tables of Feed Composition. Natl. Acad. Press, Washington, DC.

NRC. 2000. Nutrient Requirements of Beef Cattle. 7th rev. ed. Natl. Acad. Press, Washington, DC.

Olson, D. P., L. F. Woodward, R. C. Bull, and D. O. Everson. 1981. Immunoglobulin levels in serum and colostral whey of protein-metabolizable energy restricted beef cows. Res. Vet. Sci. 30:49–52.

Perino, L. J., T. E. Wittum, and G. S. Ross. 1995. Effects of various risk factors on plasma protein and serum immunoglobulin concentrations of calves at postpartum hours 10 and 24. Am. J. Vet. Res. 56:1144–1148.

Pompéia, C., L. R. Lopes, C. K. Miyasaka, J. Procópio, P. Sannomiya, and R. Curi. 2000. Effect of fatty acids on leukocyte function. Braz. J. Med. Biol. Res. 33:1255–1268.

Quigley, J. D., and J. J. Drewry. 1998. Nutrient and immunity transfer from cow to calf pre- and postcalving. J. Dairy Sci. 81:2779–2790.

Wagner, J. J., K. S. Lusby, J. W. Oltjen, J. Rakestraw, R. P. Wettemann, and L. E. Walters. 1988. Carcass composition in mature Hereford cows: Estimation and effect on daily metabolizable energy during winter. J. Anim. Sci. 66:603–612.

Whitney, M. B., B. W. Hess, J. E. Kaltenbach, H. J. Harlow, and D. C. Rule. 1999. Direct transesterification of lipids from feedstuffs and ruminal bacteria. Can. J. Anim. Sci. 79:247–249.

Wittum, T. E., and L. J. Perino. 1995. Passive immune status at postpartum hour 24 and long term health and performance of calves. Am. J. Vet. Res. 56:1149–1154.